Cigarette Smoke and Lactate Extraction. The report of (61) Summers et al. is an important one because it relates directly to the influence of cigarette smoking on patients with severe angina pectoris. It is more relevant than the observations of Aronow and Rakow discussed in page 483 (a). Lactate production of the myocardium was used by Summers as criteria for ischemia whereas exercise tolerance was used by Aronow and Rakow.

Portions of the Introduction, Discussion and Summary of (61) Summers et al. are quoted as follows:

Epidemiologic studies have demonstrated a significant relationship between cigarette smoking and obliterative vascular disease.[1] Of clinical importance is the significant relationship between cigarette smoking, myocardial infarction, and sudden death. Several studies[2-5] show an increase in deaths from coronary disease in cigarette smokers.

The objectives of this study were to determine whether the inhalation of cigarette smoke could acutely induce myocardial ischemia as reflected by abnormal myocardial lactate metabolism in patients with severe diffuse obstructive coronary artery disease, to compare this situation to the effects of catecholamine administration, and to measure some of the hemodynamic responses to the inhalation of cigarette smoke.

The absence of lactate production during smoking must be further considered. It might mean that the hemodynamic changes induced were not sufficient to cause myocardial ischemia, or that the method used was insufficiently sensitive to determine this occurrence. It would seem that the former was more likely since clinical expression of ischemia in the form of angina pectoris is not more frequently associated with smoking[7] and might be expected to be so if ischemia was produced. Another alternative would be that coronary blood flow was increased in these patients. Regan and associates[11] found no increase in coronary flow in subjects with coronary artery disease during smoking. Finally, nicotine might cause regional flow redistribution to the benefit of potentially underperfused areas. Evaluation of this possibility awaits the availability of methods for studying regional flow in the intact heart.

Summary

Fifteen patients with angiographically demonstrated severe obstructive coronary artery disease were the study subjects. Hemodynamic measurements were made before and during cigarette smoking, and metabolic evaluation was performed in these states and during isoproterenol stress. Heart rate, aortic pressure, systolic ejection and diastolic filling periods were measured. Myocardial lactate extraction was evaluated at three coronary sinus drainage sites. Despite increases in heart rate, aortic pressure, the systolic ejection period per minute, and the tension-time index per minute lactate production was not induced by smoking in any patient without lactate production in the control state. In 3 patients with lactate abnormalities prior to smoking, inhalation of cigarette smoke sustained and slightly aggravated this circumstance. Isoproterenol induced lactate production in all but 1 patient.

Cigarette smoking in a group of patients with severe obstructive coronary artery disease did not produce evidence of ischemia as determined by abnormalities in lactate extraction.

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